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NEWS 21	JAN 27	A new search aid, the Company Name Thesaurus, available in CA/CAplus
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                                                           ENTRY    SESSION
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=> s TNFR(w)DD
          1077 TNFR
            95 TNFRS
          1102 TNFR
                 (TNFR OR TNFRS)
          7892 DD
          1630 DDS
          9486 DD
                 (DD OR DDS)
L1           2 TNFR(W)DD

=> d 1-2

L1   ANSWER 1 OF 2  CAPLUS  COPYRIGHT 2004 ACS on STN
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AN 2001:190739 CAPLUS
DN 134:339415
TI Structure-activity relationship of the p55 TNF receptor death domain and its lymphoproliferation mutants
AU De Wilde, Gert; Murray-Rust, Judith; Boone, Elke; Olerenshaw, Dionne; McDonald, Neil Q.; Ibanez, Carlos; Haegeman, Guy; Wollmer, Axel; Federwisch, Matthias
CS Department of Molecular Biology, University of Gent-VIB, Belg.
SO European Journal of Biochemistry (2001), 268(5), 1382-1391
    CODEN: EJBCAI; ISSN: 0014-2956
PB Blackwell Science Ltd.
DT Journal
LA English
RE.CNT 43 THERE ARE 43 CITED REFERENCES AVAILABLE FOR THIS RECORD
            ALL CITATIONS AVAILABLE IN THE RE FORMAT

L1 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2000:492797 CAPLUS
DN 133:251048
TI Mutational analysis and NMR studies of the death domain of the tumor necrosis factor receptor-1
AU Telliez, Jean-Baptiste; Xu, Guang-Yi; Woronicz, John D.; Hsu, Sang; Wu, Jing-Lun; Lin, Laura; Sukits, Steven F.; Powers, Robert; Lin, Lih-Ling
CS Department of Musculoskeletal Science and, Wyeth Res., Cambridge, MA, USA
SO Journal of Molecular Biology (2000), 300(5), 1323-1333
    CODEN: JMOBAK; ISSN: 0022-2836
PB Academic Press
DT Journal
LA English
RE.CNT 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD
            ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> s (tumor necrosis factor death domain)
        297291 TUMOR
        122350 TUMORS
        337436 TUMOR
                  (TUMOR OR TUMORS)
         88862 NECROSIS
             2 NECROSISES
         88864 NECROSIS
                  (NECROSIS OR NECROSISES)
        814930 FACTOR
        717946 FACTORS
       1288576 FACTOR
                  (FACTOR OR FACTORS)
        105566 DEATH
          8785 DEATHS
        111910 DEATH
                  (DEATH OR DEATHS)
        217943 DOMAIN
        116303 DOMAINS
        275587 DOMAIN
                  (DOMAIN OR DOMAINS)
L2   6 (TUMOR NECROSIS FACTOR DEATH DOMAIN)
                  (TUMOR(W)NECROSIS(W)FACTOR(W)DEATH(W)DOMAIN)

=> d bib, abs 1-6

L2 ANSWER 1 OF 6 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2003:886061 CAPLUS
DN 139:349546
TI The death domain kinase RIP1 is essential for tumor necrosis factor alpha signaling to p38 mitogen-activated protein kinase

AU Lee, Thomas H.; Huang, Qiaojia; Oikemus, Sarah; Shank, Jennifer; Ventura, Juan-jose; Cusson, Nicole; Vaillancourt, Richard R.; Su, Bing; Davis, Roger J.; Kelliher, Michelle A.
CS Department of Cancer Biology and Interdisciplinary Graduate Program, University of Massachusetts Medical School, Worcester, MA, USA
SO Molecular and Cellular Biology (2003), 23(22), 8377-8385
CODEN: MCEBD4; ISSN: 0270-7306
PB American Society for Microbiology
DT Journal
LA English
AB The cytokine tumor necrosis factor alpha (TNF-α) stimulates the NF-κB, SAPK/JNK, and p38 mitogen-activated protein (MAP) kinase pathways by recruiting RIP1 and TRAF2 proteins to the tumor necrosis factor receptor 1 (TNFR1). Genetic studies have revealed that RIP1 links the TNFR1 to the IκB kinase (IKK) complex, whereas TRAF2 couples the TNFR1 to the SAPK/JNK cascade. In transfection studies, RIP1 and TRAF2 stimulate p38 MAP kinase activation, and dominant-neg. forms of RIP1 and TRAF2 inhibit TNF-α-induced p38 MAP kinase activation. We found TNF-α-induced p38 MAP kinase activation and interleukin-6 (IL-6) production impaired in rip1-/- murine embryonic fibroblasts (MEF) but unaffected in traf2-/- MEF. Yet, both rip1-/- and traf2-/- MEF exhibit a normal p38 MAP kinase response to inducers of osmotic shock or IL-1α. Thus, RIP1 is a specific mediator of the p38 MAP kinase response to TNF-α. These studies suggest that TNF-α-induced activation of p38 MAP kinase and SAPK/JNK pathways bifurcate at the level of RIP1 and TRAF2. Moreover, endogenous RIP1 assocs. with the MAP kinase kinase kinase (MAP3K) MEKK3 in TNF-α-treated cells, and decreased TNF-α-induced p38 MAP kinase activation is observed in Mekk3-/- cells. These studies suggest a mechanism whereby RIP1 may mediate the p38 MAP kinase response to TNF-α, by recruiting the MAP3K MEKK3.
RE.CNT 63 THERE ARE 63 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 2 OF 6 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2002:537202 CAPLUS
DN 137:260598
TI The death domain of NF-κB1 p105 is essential for signal-induced p105 proteolysis
AU Beinke, Soren; Belich, Monica P.; Ley, Steven C.
CS Division of Immune Cell Biology, National Institute for Medical Research, London, NW7 1AA, UK
SO Journal of Biological Chemistry (2002), 277(27), 24162-24168
CODEN: JBCHA3; ISSN: 0021-9258
PB American Society for Biochemistry and Molecular Biology
DT Journal
LA English
AB Stimulation of cells with tumor necrosis factor α (TNFα) triggers NF-κB1 p105 proteolysis, releasing associated Rel subunits to translocate into the nucleus and modulate target gene expression. Phosphorylation of serine 927 within the p105 PEST region by the IκB kinase (IKK) complex is required to promote p105 proteolysis in response to TNFα stimulation. In this study, the role of the p105 death domain (DD) in signal-induced p105 proteolysis is investigated. Endogenous p105 is shown to interact with the IKK complex in HeLa cells, and transient transfection expts. in 293 cells indicate that each of the catalytic components of the IKK complex, IKK1 and IKK2, can bind to p105. Interaction of p105 with both IKK1 and IKK2 is substantially reduced by deletion of the p105 DD or introduction of a specific point mutation (L841A) into the p105 DD homologous to the lpr mutation in Fas. Phosphorylation of immunopptd. p105 on serine 927 by purified recombinant IKK1 or IKK2 protein in vitro is dramatically reduced in both DD mutants relative to wild type. Furthermore, both of the DD mutations significantly impair the ability of low concns. of IKK2 to induce p105 serine 927 phosphorylation and proteolysis in transiently transfected 3T3

cells. However, high levels of transiently expressed IKK2 bypass the requirement for the p105 DD to induce p105 serine 927 phosphorylation. Finally, p105 serine 927 phosphorylation by the endogenous IKK complex after TNFα stimulation and subsequent p105 proteolysis is blocked in both p105 DD mutants when stably expressed in HeLa cells. Thus, the p105 DD acts as a docking site for IKK, increasing its local concentration in the vicinity of the p105 PEST region and facilitating efficient serine 927 phosphorylation.

RE.CNT 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 3 OF 6 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2001:386764 CAPLUS
DN 135:240479
TI Death domain signaling and its role in the central nervous system
AU Bruce-Keller, Annadora J.
CS Dep. Anatomy and Neurobiol., Univ. Kentucky, Lexington, KY, 40536-0298, USA
SO Advances in Cell Aging and Gerontology (2001), 5(Programmed Cell Death, Volume I), 39-65
CODEN: ACAGF5
PB Elsevier Science B.V.
DT Journal; General Review
LA English
AB A review with many refs. summarizes the different death receptors and the intracellular pathways they activate to induce cell life or death. The cloning and characterization of death receptors and their myriad of control mechanisms, and their roles in human physiol. and pathophysiol. are described. Topics discussed include death domain signaling components; initiation and execution of the death signal; death receptors in the central nervous system; alternative death receptor signaling; potent neuroprotective properties of tumor necrosis factor; apoptosis inhibitors; and NF-κB activation.

RE.CNT 148 THERE ARE 148 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 4 OF 6 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2000:790340 CAPLUS
DN 133:355211
TI Death domain-containing receptor 5 and compns. for treatment of immunity-related diseases, viral diseases, and cancer
IN Ni, Jian; Gentz, Reiner L.; Yu, Guo-liang; Rosen, Craig A.
PA Human Genome Sciences, Inc., USA
SO PCT Int. Appl., 266 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 2

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2000066156	A1	20001109	WO 2000-US12041	20000504
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1196191	A1	20020417	EP 2000-930329	20000504
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
JP 2002543151	T2	20021217	JP 2000-615040	20000504

	US 2002072091	A1	20020613	US 2001-874138	20010606
PRAI	US 1999-132498P	P	19990504		
	US 1999-133238P	P	19990507		
	US 1999-148939P	P	19990813		
	US 1997-40846P	P	19970317		
	US 1997-54021P	P	19970729		
	US 1998-42583	A1	19980317		
	US 2000-565009	A1	20000504		
	WO 2000-US12041	W	20000504		

AB The present invention relates to novel Death Domain Containing Receptor-5 (DR5) proteins which are members of the tumor necrosis factor (TNF) receptor family, and have now been shown to bind TRAIL. In particular, isolated nucleic acid mols. are provided encoding the human DR5 proteins. DR5 polypeptides are also provided as are vectors, host cells and recombinant methods for producing the same. The invention further relates to screening methods for identifying agonists and antagonists of DR5 activity, e.g., for treating graft-vs.-host disease, viral infection, cancer, and immune diseases.

RE.CNT 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 5 OF 6 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2000:617304 CAPLUS
DN 134:177079
TI The death domain of tumor necrosis factor receptor 1 is necessary but not sufficient for Golgi retention of the receptor and mediates receptor desensitization
AU Gaeta, Mary Lou; Johnson, David R.; Kluger, Martin S.; Pober, Jordan S.
CS Department of Pediatrics, Yale University School of Medicine, New Haven, CT, 06510, USA
SO Laboratory Investigation (2000), 80(8), 1185-1194
CODEN: LAINAW; ISSN: 0023-6837
PB Lippincott Williams & Wilkins
DT Journal
LA English
AB TNF signals are mediated through two different receptors, TNFR1 and TNFR2. In endothelial cells, TNFR1 is predominantly localized in the Golgi apparatus and TNFR2 on the plasma membrane. To investigate structural features responsible for the disparate localization, endothelial cells were transfected with epitope-tagged or green fluorescent protein-fused wild type and mutant receptor mols. Wild type receptors recapitulated the distribution of endogenous receptors. Deletions of the entire TNFR1 intracellular domain or of the C-terminal death domain (TNFR1-DD) allowed expression of the receptor on the plasma membrane. However, addition of the death domain to the C-terminus of TNFR2 (TNFR2+DD) did not lead to Golgi-retention of this chimeric receptor. Overexpressed TNFR1, TNFR2, and TNFR2+DD increased basal expression of a cotransfected NF-kB-dependent promoter-reporter gene. Overexpressed TNFR1-DD did not activate NF-kB but acted as a ligand-specific dominant neg. inhibitor of TNF actions. Unexpectedly, TNF responses were also inhibited by overexpressed TNFR1 and TNFR2+DD, but not TNFR2. We conclude that the death domain of TNFR1 is required for retention of TNFR1 in the Golgi apparatus but is not sufficient to direct Golgi retention of a TNFR2+DD chimera, and that overexpressed receptors that contain the death domain (TNFR1 and TNFR2+DD) spontaneously activate NF-kB while inhibiting TNF responses.

RE.CNT 41 THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 6 OF 6 CAPLUS COPYRIGHT 2004 ACS on STN
AN 1998:215327 CAPLUS
DN 129:3726
TI The death domain kinase RIP mediates the TNF-induced NF-κB signal
AU Kelliher, Michelle A.; Grimm, Stefan; Ishida, Yasumasa; Kuo, Frank; Stanger, Ben Z.; Leder, Philip

CS Harvard Med. Sch., Howard Hughes Med. Inst., Boston, MA, 02115, USA
SO Immunity (1998), 8(3), 297-303
CODEN: IUNIEH; ISSN: 1074-7613
PB Cell Press
DT Journal
LA English
AB The death domain serine/threonine kinase RIP interacts with the death receptors Fas and tumor necrosis receptor 1 (TNFR1). In vitro, RIP stimulates apoptosis, SAPK/JNK, and NF-κB activation. To define the physiol. role(s) that RIP plays in regulating apoptosis in vivo, the authors introduced a rip null mutation in mice through homologous recombination. RIP-deficient mice appear normal at birth but fail to thrive, displaying extensive apoptosis in both the lymphoid and adipose tissue and dying at 1-3 days of age. In contrast to a normal thymic anti-Fas response, rip-/- cells are highly sensitive to TNFα-induced cell death. Sensitivity to TNFα-mediated cell death in rip-/- cells is accompanied by a failure to activate the transcription factor NF-κB.
RE.CNT 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

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